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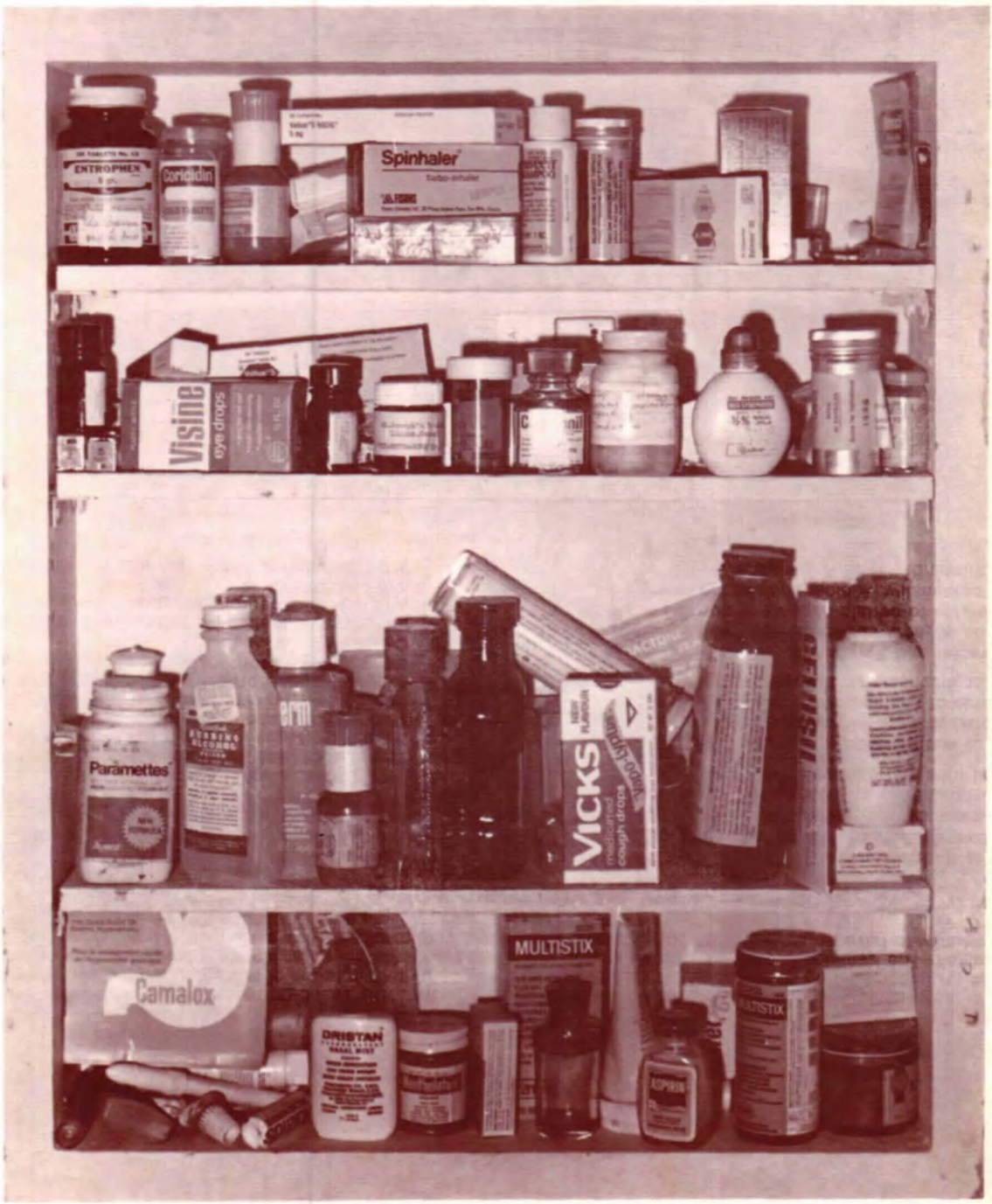
May - Vol. 46, No. 4

The University of Western Ontario

Medical Journal



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May 1976

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Guest Editorial

The Human Dimension in Medical Education

John Biehn, M.D.,
Department of Family Medicine, U.W.O.

In talking with medical students, and recalling my own experiences as a student, I am impressed that many of the feelings experienced by students today are remarkably similar to those which bothered me and which I had little opportunity to express, nor the willingness, insight and ability to articulate at the time. I am inclined to believe that these feelings tend to be present in a majority of students to varying degrees, and are not dealt with constructively, either by the Faculty or themselves. What effect this may have on the quality of medical education is a matter for debate, but I would surmise that the effect might tend to be more negative than positive in a learning sense.

In attempting to define some of these feelings and issues, it is not my intent to criticize my own Faculty, for which I have the utmost respect and regard, because I see my behaviour as a teacher inadvertently and without conscious intent contributing to some of these problems. However, having had a recent opportunity to engage in dialogue on this whole matter, I feel it would be worthwhile to share my observations, realizing that what I say will not be accepted by or seem very relevant to some students and faculty members. I would be gratified if my observations struck a responsive chord in only a few.

I recall with considerable clarity the overriding emotions of fear, anxiety, insecurity and frustration which were constant during my undergraduate education. I am persuaded to believe that these feelings are present in most medical students, and are not openly discussed for two major reasons. Firstly, many students believe that such feelings are personally unique. Secondly, to admit that these feelings operate at whatever level is subjectively interpreted to indicate weakness and perhaps an inappropriate career choice, and most of us have a need to supress such disturbing thoughts. And yet most physicians, on deep reflection, will admit that at least as an undergraduate student if not indeed at later times, there were moments of despair, perhaps numerous ones, when the thought of dropping out was seriously entertained. It is of considerable interest to me why this should be so. Though there are likely many reasons, I feel there are some common threads which run through this dilemma.

The majority of students enter medicine from a background of science and mathematics, which tends to persuade one that there are absolute answers to problems, and that such absolutes can be achieved and realized through intense study, as witnessed by sucess in obtaining admission to Medical School. It is unreasonable to suppose that students do not approach the study of medicine from the same perspective. The emotions of fear and anxiety are likely to result from the early realization that, given the volume of material to assimilate, the task is one of enormous magnitude. Such feelings are undoubtedly underscored and magnified by teachers in any discipline who persuasively advance the argument that a detailed knowledge of their discipline is essential to the adequate functioning of physicians in all fields of endeavour. Experience would indicate that this is not necessarily the case, but the student unfortunately is in no position to realize this, or to seriously doubt the veracity of his or her teacher.

At some stage the student begins to realize that there are finite limits to his or her ability to assimilate all the factual material which is thrust their way. My realization of this finiteness induced in me a tremendous feeling of insecurity which, in all honesty, I continue to struggle with. In the clinical disciplines, this feeling was further compounded by my increasing awareness that there were and are very few absolutes. One could study in detail a specific disease entity and its treatment, only to discover that in a particular patient all the criteria for diagnosis were rarely met, and if they were, the studied treatment did not totally apply to a particular patient.

I can vividly recall and continue to experience a tremendous sense of frustration in these circumstances. I have reason to believe that many students can identify such feelings in themselves. A common reaction to these frustrations and other feelings is to project the cause as being a failure in medical education, which in a very real sense it is. I believe that the continued prevalence of "bitch sessions" among students, and not infrequent confrontations with faculty, represents the outward manifestations of these deeply rooted, yet unexpressed feelings of fear, anxiety, insecurity and frustration.

I see myself slowly but steadily evolving a position which, briefly stated, tends to an increasing conviction that for myself at any rate, the key to increasing effectiveness as a physician and teacher is my continued growth as a person and my understanding of that growth process. It

pains me considerably to realize the extent to which this process was neglected and subdued as a student, both by the medical education process and particularly by myself. For this deficiency no blame is attached, because I feel we are only beginning to understand the complexities and subtle nuances of human behaviour. I believe strongly that these evolving understandings, however inexactly, must be applied to the process of medical education so that students can at least begin to realize, and in some way come to understand what is happening to them and why it is happening as they pursue a degree in a richly rewarding profession. We pay a great price if, in this pursuit, we deny our humanity.

One might well ask how such a process can begin to take place. There is no single answer of course. Faculty however can acknowledge to students that effective functioning as a physician is dependent on many things other than detailed factual knowledge, and at the same time, indicate a willingness to recognize and accept the feelings of students as normal and understandable. Students on the other hand can begin to come to an awareness that the problems they experience are not entirely the fault of Faculty, but largely part of the process in their own growth and development as persons and physicians. The processes and pathways through which such mutually advantageous steps can be taken are only limited by the imagination. Though I have a healthy scepticism towards the effectiveness of small group discussion as a useful tool in dealing with these problems, I have had personal experience which leads me to feel that they can exert a very positive influence.

I do not mean to imply that any innovations along these lines would simplify the educational process. Indeed, another dimension would be introduced: the human dimension. It would be wrong to deny the need for a competent physician to learn a core of basic knowledge as an undergraduate student. But we can negate the myth that the half life of medical knowledge is five years, or whatever number of years is presently in vogue, and suggest realistically that continued effectiveness will depend as much on such factors as judgement, understanding and compassion for the patient, as it does on knowledge. We can emphasize that medical errors are more dependent on these factors than lack of factual knowledge. As well, we can empathize with the students as colleagues in their struggles to grow and develop. When placed in such perspective, it seems to me that the process of medical education is potentially more exciting, challenging and rewarding.

Acknowledgement

In February 1976, I had the good fortune to attend a conference on "Human Dimensions in Medical Education". This allowed me an opportunity to reflect deeply on the philosophy and process of medical education and to clarify some of my rather hazy thinking in this area. I would highly recommend future conferences to any interested students or faculty members.

Information can be obtained by writing:

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Point of View

Corla LeMorrive

A Layperson's Plea for "Family" Medicine

It was hard enough being a teenager twenty years ago when I was growing up, but now it must be much worse. A scant generation ago teenagers were governed by norms of social behaviour. We worried about our clothes, our popularity and our complexions; we admired success and aspired with pride to the middle class. Although we may have fought with our parents, we bowed to their authority. In the intervening years the rules we often hated (but accepted and certainly needed) have apparently evaporated. Teenagers today are assaulted by an insidious counter-culture espousing anti-heroes and evoking a disenchantment with all facets of society. Our naive flamboyancy has been replaced by doubt, despondency and a vague malaise which seems to shroud the mystical teenage years. Trained at school to think independently and challenge even the most ordinary rules and ideas, teenagers languish in the terrifying situation of having to survive without the security of an established set of social mores. When forced to construct an individual moral code amidst potentially lethal temptations, the weaker ones often fail the test.

Not surprisingly, many parents are panic-stricken. The rules that applied a generation ago are ludicrously obsolete, and when confronted with teenage indulgence in drugs, promiscuity and disrespect for established rules, they freeze. Parents live in fear of the morass waiting to swallow their children. At the same time, guilt-ridden for harbouring such emotions, they tend to over-compensate by being too understanding, too lenient, too reluctant to make unpopular but essential rules. In short, they feel impotent.

In our family we were forced to face the issue head-on. Our fifteen-year old daughter began to experiment with liquor and marijuana, to disrespect every facet of her home life, and to lie to cover up her unacceptable behaviour and emotions. Like most parents confronted with a "rebellious child", we dealth with what we could and silently worried about the rest. But our relationship in general continued to deteriorate, and I sought the advice of a family psychologist when the dimensions of the problem became evident. Meanwhile our daughter became entrenched in her contemptuous attitude for her family and home. Finally, violating modern liberal concepts of the sacrosanct privacy of the individual, my husband (my daughter's step-father) took the desperate step of reading her diary.

The diary not only revealed that she had been helping herself to his pocket-money to buy marijuana and had been associating with a fast crowd in a forbidden area of town, but it also seemed to lay bare something far more serious. She indicated in its secret pages a contempt, even hatred, of her parents. Page after page was packed with venom towards those with whom she lived. My husband was deeply upset by what he read and showed the diary to me. I had just read a similar diary, **Go Ask Alice**, that my daughter had also read and found very moving. There were many similarities between the two pieces of work and what she wrote might well have been largely fictitious. I suggested that we keep a close watch but avoid a hasty decision.

Weeks later the volatile situation exploded. A burst of totally unwarranted and unacceptable behaviour convinced me that it was time to act upon the advice of the psychologist. My daughter had often expressed a wish to live with her father. Now, when life was intolerable in my home, we acquiesced in her request, and sent her to her other parent in another city. Her father, always a competent parent, did his best to help her. But he had a new, young wife and two other children in his home and the task was too difficult. She began to run away and display as much contempt for him as she had earlier shown to me. To preserve himself and his family, he was forced to send her back.

Upon her return we began to see a youth counsellor – a qualified medical doctor operating under OHIP out of a local hospital clinic – who diagnosed that she was spoiled and simply needed firm discipline. Although I doubted such a simplistic judgment I followed the counsellor's suggestions. Inside of a month, however, the situation had again reached a crisis. My daughter seemed unable to cope with the most minor family responsibilities. She conceived elaborate tales to conceal her involvement with a group of older boys who took her joy-riding in stolen cars. We realized that we were naively applying band-aids while the child was bleeding to death.

We told the youth counsellor that more intensive therapy was necessary if our daughter was to be helped, and suggested that her recent actions were signs that she was crying out for assistance. But the counsellor bluntly questioned our judgement and our competence, referred us to the Children's Aid Society and a child psychiatrist, and withdrew from the case. A Children's Aid Society emergency worker subsequently proposed that our daughter spend some time in a foster home to provide a mutual cooling-off period. We agreed to a temporary, non-ward arrangement with the CAS, and waited a few days until a home could be made available. But before action could be taken, the police telephoned early one morning to report that our daughter had been picked up in a stolen car. She had spend several hours in a cell. I was overwhelmed with the realization of my total inability to provide the kind of environment she required.

The acceptance of this situation was perhaps the most difficult thing I have ever been called upon to do. Young people, we are told, do not become sick by themselves. They are products of their surroundings. The mother traditionally provides the greatest single influence in their lives. Therefore, it is only reasonable and logical to deduce that the mother is culpable for the emotional disturbances of her child. A mother is a sensitive creature, taught by centuries of motherhood to respond to the needs and wants of her family as her own means of fulfillment. Thus the failure, weaknesses and problems, as well as the successes and accomplishments of her family become hers also. The burden upon the mental health of a mother who is forced to recognize that her child requires treatment for emotional problems is extreme. In both society's eyes and her own, she is to blame. Little wonder that parents are slow to acknowledge that their child needs professional help; little wonder that they bury their heads in the sand until it is too late for all but the drastic treatment. Little wonder that they are guilt-ridden and ashamed.

But why are professionals just as reluctant or unable to advise proper treatment? The family psychologist I had seen months earlier never talked to my daughter, nor did he suggest that she might need counselling. My family physician a few months later (without

seeing my daughter) refused to refer her for professional help on the grounds that such a move would look as though we thought she was mentally ill! The youth counsellor never did admit that this young teenager's problems had spiralled beyond his capability and training.

In the end it was I, her mother, who forced the issue, alienating the youth counsellor by demanding a psychological assessment. I also had to admit defeat by calling upon the Children's Aid Society and placing her in their non-ward care. I was perhaps the person most responsible for her appearing before the juvenile court as a child in need of protection (as such children are considered by current court procedures).

It was a humiliating experience to appear before a judge and reveal the reasons for our daughter requiring protection (not help, but protection). In retrospect, however, the judge of the juvenile court succeeded where trained medical people had not. He followed the recommendation of the Children's Aid Society that she be placed in a foster home for observation. In the meantime he ordered a complete psychological assessment. We appeared before the bench a month later. By this time the foster parents had recognized that her problems were too serious to be dealt with in a family environment and the psychiatrist had also recommended that she be placed in a treatment centre for emotionally disturbed adolescents.

One would have thought at this point that the preliminaries were over. On the contrary, they were only beginning. The Children's Aid Society made its own assessment and unilaterally decided to place her in an assessment centre for further observation and recommendation. At the end of two weeks the centre reached the same conclusion as the psychiatrist had arrived at earlier. In the meantime we began to search out the implications of the psychiatric report. What exactly was a treatment centre? Were they all similar in methodology and treatment? Were they all equally good? What about waiting lists? Information about these relatively simple questions is very difficult to obtain. The psychiatrist, for example, was unable or unwilling to give us any advice. After sleuthing in many places and by many means we came to the conclusion that one of the best centres in the province was in the city where we lived.

Once the Children's Aid Society made its assessment they approached us about treatment facilities and recommended their own. When we said both innocently and candidly that we preferred to place her in another facility, we were totally unprepared for the hostile reaction our words apparently provoked. We were alternately bullied, threatened with legal action and assaulted with statments that verged on being outright lies. We were told, notwithstanding at least two psychiatric assessments to the contrary, that our daughter did not need professional care; she needed only a reasonable home life, love and compentent care. Although the CAS could do nothing legally to prevent our request, they employed the only tactic at their disposal – procrastination. By this means proceedings could be delayed if not prevented. Meanwhile, while we fought to obtain proper care for our daughter, a murder-suicide case involving a disturbed teenage boy hit the newspapers. In the midst of the controversy the coroner of Ontario and the media castigated the boy's parents for ignoring signs of his illness (one of which was a diary), and for failing to obtain proper professional guidance. The total situation was too tragic to be considered ironic.

Six weeks passed while the Children's Aid Society did little if anything – the request-for-admission forms lay unsigned, the questionnaires and case history unanswered. Despairing that the needs of a sick child were apparently being subordinated to bureaucratic jealousy, we approached our most sympathetic ally in the case – the juvenile court judge. He recognized immediately both the nature of the problem and how to solve it. He also supported wholeheartedly our choice of a treatment centre, reiterating that he had found it to be one of the best in the province. We left feeling elated.

When the judge applied the pressure at his discretion the wheels again began to roll. In another six weeks our daughter was admitted for treatment. The experience from start to finish had been harrowing. Everyone, including me, seemed to blame me for what had happened - the psychologist, my family doctor, the youth counsellor, a tactless police officer, even the Children's Aid Society. Only a probation officer and the judge showed any signs of believing that the case was not a question of guilt or innocence to be pronounced upon a home life – it was a question of helping a disturbed child. In all, we spent almost a year and a half searching for help, and we were involved with psychologists, psychiatrists, a youth counsellor, our family doctor, the courts and the Children's Aid Society. Not only was our search a tremendous drain on our time and energies and the resources provided by tax dollars, but we cannot help but feel that many of her problems could have been avoided, had she received prompt and proper attention at the outset of her difficulties.

Her admission for residential treatment has not brought an end to our problems. My daughter believes that we have cast her out, rejected her; and she refuses to have anything to do with us. She hates the centre and phones occasionally to tell us how callous and cruel we are to force her to stay in a cesspool where people try to break the walls down and where the floors are knee-deep in dirt. She wants out, she cries; she screams that we are messed up, not she; we are to blame and are punishing her for something she did not do. Although we know that what she says is untrue, it is hard to react calmly to her heart-rending appeals. It is hard to cast off the guilt when others put it back and to remain steadfast and confident that we are doing and have done the right thing when there is no outside support for our actions. It is difficult, even mentally exhausting. But perhaps the worst is over. And the most important thing is that she is under proper care.

It is, however, my turn to appeal. My experiences seem to have demonstrated that we desperately need the establishment of an entirely new machinery for detecting and treating children with emotional problems, before the child shoots someone or destroys his own life. To rely upon parents to take the initiative when society makes them the guilty parties is to ask the impossible. Many competent, informed psychiatrists and family doctors are badly needed, and they require the training not only to diagnose an illness, but also to advise upon proper care and treatment. Facilities available for testing, assessing and treating are sorely lacking. And counselling for parents who are wondering whether their child is abnormal or whether they themselves are simply paranoid or over-protective are non-existent. Even where facilities like treatment centres exist, they seem to be competitive rather than complementary. We are continually told that proper, stream-lined facilities would be too costly. But can there be a better demonstration than our experience that present community resources are poorly organized and inefficient, that we are caught in a bureaucratic jungle of red-tape, conflicting jurisdictions, competing forms of treatment, and professional jealousies. Raising a family is perhaps the most difficult task of a life-time, yet it is doubtless the one for which we are the least prepared, the least counselled, and the least supported. How much easier it would be to help those who need help if the facilities to do so quickly and decisively could be established.

Medical Imagination

It Happened in Canadian Medicine
"Mail-order Medicine" in the Western Fur Trade

Where ranches now sprawl in checkerboard fashion on the Canadian prairie, fur traders once wandered free. To those of us locked in the iron jaw of the urban work ethic, the fur trader's existence is often romantically alluring. We envy the uncomplicated vigour with which he challenged the elements of nature for survival, living out his natural span of life in harmony with his primeval world.

In truth the fur trader's rigorous battles were romantic only in retrospect. He frequently endured an unvarying menu of perhaps fish, flour, and pemmican, with few vegetables for months on end. Isolation and loneliness were the scourge of the wilderness. A person could die alone and rot undiscovered for months; he could be sick, unable to reach a doctor, and lie in unnecessary agony.

Nineteenth century fur trade correspondence is laced with descriptions of just such situations. Illness was a major factor of fur trade life, and rare indeed was the letter not reporting incapacitation or death. Perhaps to present-day observers the strangest letters are those to doctors requesting medication. The mail might take from three weeks to six months to reach the physician and just as long for the medication to return. The requests might be pathetic, tragic, or even amusing.

A few examples are offered for the edification of the modern reader. A note James Hargrave, a chief trader at York Factory, sent to Robert Wilson at Severn in 1835 evokes an image of either a dying man or an incurable hypochondriac: "I send you a few medicines from the Doctor for your Sore feet & ears, but I regret he cannot prescribe for the pain in your breast without a fuller description of your case than what you gave me." But to the twentieth century observer perhaps a doctor's sage advice to a young and constipated fur trader best captures the imagination:

York Factory
15th February, 1847

To Mr. W. Lane

Sir:

I herewith send vou a box of compound Colocynth Pills according to your request. I would not recommend to you to indulge very frequently in them as the continued use of any laxative medicine is apt to diminish the natural vigour of the bowels and thus render the necessity more confirmed. I would advise you rather occasionally to use injections of cold water which has a beneficial effect in confirming the tone of the more immediately expellent portion of the internal canal. I remain

W. Smellie, M.D.

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Hypochondria in General Practice

Mark Leith

Definition

According to the Diagnostic and Statistical Manual II of the American Medical Association hypochondrial neurosis is defined as the following.

> Hypochondrial neurosis is dominated by preoccupation of the body and with the fear of presumed diseases of various organs. Though the fears are not of delusional quality as in psychotic depressions, it persists despite reassurance. The condition differs from hysterical neurosis in that there are no actual losses or distortions of function.

In terms of general practice this patient may be more aptly defined as the one who seems to refuse to get better, the patient one would rather not see, the patient presenting with multiple vague symptoms, the patient who seems overly concerned about his health, the patient with a history of multiple operations and multiple doctors. This above described patient may be an end-product of a process I will describe later on in this paper.

Histo. y

The word hypochondria comes from the Greek, 'hypo' meaning under and 'chondria' meaning chest. Thus, hypochondria was felt to be a physical ailment originating in the upper abdominal region. Writings taken from the 17th century continued to describe hypochondria as a physical ailment: "sharp belchings, fullsome gratuities, heat in the bowels, wind and grumblings in the gut, vehement gripings...". It was felt to be the male version of hysteria, hysteria arising from vapours in the uterus, hypochondria arising from vapours in the spleen. In the 17th century attempts were made by the philosopher Burton to combine the gastoentrological with the psychological aspects of this disease and he thus coined the phrase 'flatuous melancholy'. The dualism between the psychological and physical etiology of this disease continued until the time of Freud. Freud saw hypochondria as an 'actual' neurosis in which sexual energy was transformed into somatic changes. This theory will be discussed further later on. The importance of the theory rests in the fact that it introduces the concept of an unconscious psychogenic origin.

Epidemiology

A study by Keenan between 1951 and 1960 at Maudsley-Bethlem-Royal Hospital of over 500 patients estimated the incidence of hypochondria at 1 percent. He found that over 50 percent of the patients came from social class 3 as opposed to the popular conception of hypochondria being a neurosis of the upper classes. Furthermore, hypochondria was found to be equally distributed between the sexes, not predominant in females.

In a study of general practitioners in England in 1966 by Sheppard patients with psychological problems were found to present more often to their family doctors with a wider variety of complaints than controls. These complaints were especially high in the gastroentrological, the genito-urinary, and the respiratory systems.

Dynamics - Patient

We have covered previously some of the theories up until the time of Freud which brings us to a description of Freud's actual neurosis. Freud felt that in the actual neurosis the libido was blocked by anxiety and guilt towards the external object and was thus turned towards the self "Erotogencity is a property common to all organs". Thus according to actual neurosis all organs become genitalized, resulting in somatic complaints. Though this theory is limited by its strong sexual bias it does point out that hypochondrial neurosis may arise from unconscious conflicts or problems resulting in anxiety and-or guilt, which in turn, produces these complaints.

Later theories by Sullivan saw hypochondria as a protective "substitute activity" against low self-esteem and by Wahl as a defense against guilt over destructive wishes by damaging the self. Thus we see that the theories are moving away from purely sexual etiology. However neither of these above theories are all encompassing. Both I believe are examples of the concept of gain, which I intend to discuss later on in this paper.

However, before discussing the concept of gain I would like to discuss some of the observed dynamics seen in hypochondria. The first of these is denial. If a physician attempts to ask about conflicts outside of the presenting somatic complaints the patient will probably imply that everything is okay and be very resistive to further probing and insight. Patients also show regression and dependency. They turn away from threatening situations and become egocentric and self-preoccupied, particularly with normal body sensations. For example the patient may present with an odd sensation in his neck which may turn out to be his normal carotid pulse. These two defenses, regression and dependency are a part of the normal sick role. Lastly hypochondria is seen as a reaction to stress, for example following trauma, surgery, or a death in the family. From the above dynamics we see the following pattern. All of these are defenses against a threatened self, a defense against anxiety originating from within but being manifested as an anxiety about health. This results in little subjective concern about the patient's own emotional and psychological state. However attempts to make this interpretation to the patient will be resisted since one is attacking these very defenses. It will be perceived by the patient as saying "I do not believe you, it is all in your head".

This brings us to the concept of gain. Gain may be divided into two aspects - primary and secondary.

Primary gain operates on an intra-personal level, that is within the person. It is an attempt by the patient to handle conflicts and problems from within. These conflicts are usually of an aggressive or a sexual nature. Gain then serves to deny these problems, to repress possibly threatening impulses and emotions, and finally to punish for guilt arising from these conflicts by being sick.

Secondary gain operates on the interpersonal level, that is between the patient and other significant persons. It is this aspect of the neurosis that most annoys the family and the doctor. By being "sick" it allows the patient to manipulate others. it gives him license to be dependent on others, to be immature, to avoid responsibility, to receive pity, sympathy, and to avoid unpleasant and threatening social situations. The important thing to remember about both of these gains, particularly secondary gain, is that they operate on the unconscious level. What seems so obvious to the doctor is completely out of the conscious awareness of the patient. This results in frustration in the doctor and anger towards the patient

which in turn is met by anger in the patient towards their doctor.

Differential Diagnosis

..Having examined some of the dynamics which go on in the patient, it may be a good point to look at the differential diagnosis of hypochondria. First of all hypochondria differs from the malingering patient in that the malinger sits down and makes a very conscious effort to play the sick role. It differs from psycho-somatic illnesses in that psycho-somatic illnesses, for example an ulcer, the concern is appropriate to the amount of pathology. Hypochondria may be differentiated from hysterical neurosis in two ways. In hypochondria the complaints are vague and multiple whereas in hysteria the complaints are usually single and with a definitive loss of function, for example hysterical blindness. Furthermore there is a difference in affect. In hypochondria the patient is extremely anxious whereas in hysterical neurosis the patient exhibits "la belle indifference", i.e., seems not particularly concerned about the apparent loss of function.

Dynamics between the Doctor and the Patients

The following is a case taken from Dr. Balint's book, **The Doctor, the Patient and the Illness.**

Case: Mr. U, Age 36, described by Dr. E. A highly skilled worker, earning about 15£ per week, married, two children. Very happy, apart from the fact that the younger boy, four years ago, had acute nephritis, and has been ill since. Mr. U had polio as a boy and his left leg is about 4 inches shorter, requiring a boot. He deals with his infirmity quite well, however. The boy's illness is rather tragic, but he copes with it quite well, although the illness gets his wife down. He runs a car, and takes his wife out for weekends.

In February, while he was at work, someone tampered with an electrical connection and he got a very severe shock. He was thrown clear, and was out for about 15 minutes. He came around and recovered completely. I think he then saw the doctor at the works or perhaps they sent him to the casualty department at the local hospital. Two or three weeks ago he came to see me, complaining of pain all over his chest, the lower part of his back, right leg and right hand and saying that the pains were getting worse and worse. I examined him thoroughly and came to the conclusion that no organic damage had been done, although he thought something had happened to him through the electric shock. He seemed rather worried aobut it, I suggested that I get a specialist's opinion which he accepted. He came to see me again last night. He had had all the examinations. The letter from the hospital said that they could not find anything wrong and that "We would like the patient to receive a sedative". I told him that nothing wrong had been found, and he said that that was funny because his pains were much worse. He said "they seem to think that I am imagining this - I know what I have got". After talking a few minutes in a very pleasant manner he said he thought that the hospital might have made a mistake. He is definitely ill and would like to know what condition could be causing all these pains. "What does the book say about it?" I did not answer, except to say that it was not a matter of looking up the books, that for example if he had a broken leg he would not ask me the cause of it, he would ask me to get it better. Finally I said that as he could not accept the view of the hospital, would he like to go to an entirely different hospital to be examined again? He was not keen, saying that they would do the same thing - he would not find wrong. He is not sure how to get on. What was the next step, so I suggested that he should come back to the discussion in a week's time.

This case illustrates a number of the dynamics and problems involved in the doctor-patient relationship and the patients presenting with psychological problems manifested as somatic complaints. According to Balint the patient proposes, (in this case pain all over the body, except the left leg,) **an illness.** This gives the doctor two options: to treat the patient or to reject the offer. If the doctor chooses to reject the offer there is no comprimise made and this may result in hostility and decrease the compliance by the patient. If the doctor chooses to accept the illness comprimise is reached and the illness becomes, according to Balint "organized". All patient initially present "unorganized". That is they offer various illnesses until the doctor and patient can agree on accepting one of them. They thus settle down to one or two of these proposed illnesses and get "organized".

Thus an early hypochondriac may present with multiple ills to multiple doctors and over a period of time will settle down to one or two ills with one or two specific doctors. In the above case Dr. E. neither accepted or rejected the patient but sent him on for further investigation following which he was "rejected" by the consulting hospital.

The second problem this case illustrates is that of the order in which to proceed. In the above case although an emotional problem was high on the index of suspicion the patient was sent to a specialist. This resulted in the patient's feeling of rejection, ("they seem to think I am imagining things"), which resulted in the beginning of a possible argument between the doctor and the patient with an ensuing decrease in the patient's compliance concerning another possible referral. This is evident that the patient that was sent away by Dr. E. was not the same patient that came back follwing the examinations.

Why did this general practitioner concern himself mainly with the organic side of the problem? Balint argues that this is an artifact of the training of the general practitioners. Most of the training of general practitioners is done by consultant-specialists, although efforts are being made at this University to counter this bias. The specialist by definition is a physician who deals in one system of the overall aspect of disease, whereas a general practitioner is in "people" medicine. This resulting difference between the one system medicine vs. people medicine results in gaps in the training of the general practitioner which have been filled with empiricism and common sense. Further Balint argues that the specialist is biased toward the physical, feeling that a missed physical diagnosis is much more dangerous than a missed psychological one. While this may be true of the specialist consultants this is not true of the general practitioner where a missed psychological diagnosis may be as important to the patient's well being as a missed physical one. To the specialist a psychological diagnosis is often one of elimination following a long series of high powered tests. For the general practitioner however psychological diagnosis must be a positive one since a long series of examinations are either unavailable or extremely costly. This training bias may be a manifestation of a larger cultural bias in which areas such as death, sex and feelings until recently were taboo topics. These are unknown, unchartered, awkward and threatening areas of human living. A by-product of this cultural bias is a lack of language to describe psychological processes. Physical labels are much more sophisticated and precise whereas a diagnosis of neurosis is a vague diagnosis, one which anyone could make. This brings us to the third problem - what to call it.

What to call it?

In the above case we can see that the patient's first concern is to have a name for his illness — "what does the book say about it?" The patient's need to know may be part of his need to be recognized, to realize that what is bothering him has bothered other people and in a sense take away his uniqueness and resulting alienation. Dr. E's example of the man with the broken leg is in a sense off the mark. In the case of the broken leg the diagnosis was obvious and therefore the patient was ready for treatment. Whereas in the case of Mr. U the diagnosis is not so obvious. A collorary of the patient's need to be recognized, is the patient's need to be taken seriously. Mr. U felt that the hospital was not taking him seriously, feeling that it was all his imagination to which he reacted with anger — "I know what I have got". Thus a diagnosis must be established before the patient is ready for treatment.

Given that a psychological diagnosis in general practice must be a positive one and given the need of the patient to know what is wrong with him, we come to what Balint calls the level of diagnosis.

Suppose Mr. U had returned in one week's time with a sore throat and then had returned the following week with abdominal pain and then had returned a number of weeks later with shoulder pain. On one hand we could make the following diagnosis of URI, possible peptic ulscer and possible muscle spasm. However on another level we can see a pattern behind these illnesses and a possible conflict behind this pattern. By altering the kind of diagnosis that is made one may change the kind of diagnostic procedures one wishes to choose which may result in a better idea of what is actually going on and a more accurate therapy. In the above case the treatment given was reassurance and a sedative. This treatment however failed. It failed because the diagnosis was true but only true to a certain extent. It was true to the extent that nothing was organically wrong but did not actually describe the real problem. We may surmise at a deeper diagnosis given the patient's history of stress concerning his son's nephritis, his own previous battle with polio and the curious fact the pain occurs everywhere except in his crippled leg.

Psychological diagnosis however, presents certain unique problems. For one, diagnosis is inextricably intertwined with treatment, since in probing psychological areas one also releases feeling, memories, "stirs up trouble". The doctor's reluctance is based on two factors. First is his bias towards the physical, as we have seen in his training and second, his increase in responsibility. Thus the "stirred up trouble" the doctor fears in the patient may actually be his own anxiety projected on the patient. Probing into psychological areas is not without risk. But then all examinations carry with them a risk versus benefit ratio. I will discuss possible ways to decrease this risk later on in this paper concerning therapy. However the benefits of a deeper diagnosis may be found in understanding the pattern behind patient visits and in allowing the doctor to be more precise and weighing the physical versus the psychological factors within the patient. In addition the benefits of a psychological diagonis allow the doctor to take a better look at himself. This allows him to deal with the feelings of inadequacy which these kinds of patients produce in doctors due to the supposed failure of their medical treatment and, secondly, allows him to look at the frustration and hostility arising from being manipulated by these patients.

This case illustrates the relationship between the general practitioner and the specialist-consultant. In the case of Dr. E and Mr. U the patient was sent to the hospital and the specialist reported back that nothing was wrong. The general practitioner, in turn, told this to the patient. This is what Balint describes as an example of the collusion of anonymity. This collusion occurs when neither medical party takes the full responsibility for decisions made. Thus the hospital does not have full responsibility since they merely do the tests and discharge the patient. Similarily, the general practitioner does not take full responsibility in that he is able to say "this is what they told me". There is a dilution of responsibility be spreading it among a group of people with the result that no one is actually in charge. This may, in fact, explain the medical history of the patient with multiple operations. The patient is referred by his general practitioner to specialist number one who, being human, has difficulty in handling this patient. He then refers this patient to specialist number two, and so on. Along the way the patient may have an operation, without anyone actually knowing the patient, without anyone actually being in charge. The patient also may be a protagonist in this scheme. He may initially be manipulative and may develop passive - aggressive behaviour in order to play one doctor off against the other.

How does this collusion happen? Balint feels the answer to this question lies again in the training of the general practitioner. The general practitioner is taught in medical school by consultant-specialist teachers and this relationship of student and consultant-specialist teacher continues after graduation. The consultant feels he must give advice, and the general practitioner, being the student, follows his teacher's advice-although he has access to facts which the consultant does not. Thus, in the above example, it was actually the general practitioner who was in a better position to decide whether anything was actually wrong with the patient. Communication between the general practitioner and the consultant was hampered with the consultant-teacher being unable to admit that he did not know and the general practitioner unable to admit that maybe he did.

The solution to this problem may result by the general practitioner accepting that ultimately he has full responsibility for the patient. He must therefore review the consultant's report in the same manner in which he would look at a lab test, as another piece of data to be evaluated and taken into consideration in the end for his final decision. In the case above, Dr. E. could have accepted the report up to the point that nothing was organically wrong but not that absolutely nothing was wrong with the patient.

Treatment

First let us take a look at what did not work. In case of Mr. U the treatments given were reassurance and a sedative. They failed to work since the correct diagnosis, i.e., what was actually going on, was not made. Nevertheless, the two commonest therapies given in hypochondria are advice and reassurance. Balint feels that these are not necessarily bad, but that they need to be aimed with greater accuracy.

What may then work? As mentioned earlier, psychological diagnosis and treatment are intertwined. In attempting to make a diagnosis one is usually starting treatment at the same time. In a history taken for physical illness, the physician is the main protagonist. He tests out various hypothesis with questions which require short descriptive answers. In psychological diagnosis, this may be appropriate for symptoms of anxiety and depression but seldom for revealing intrapsychic conflicts. In a psychological diagnosis one is acting as an antagoinst with the patient as a protagonist or source of material, i.e., one is trying to draw out and direct what is coming from the patient. The physician has at his disposal a number of tools. The first of these is an opening non-threatening question, such as "How are things going?" as opposed to "How are the nerves?" which would immediately put this patient into a defensive posture. The second tool is silence. This allows the physician to do two things. First, it puts pressure on the patient to talk. Secondly, it allows what is in the patient's mind to come according to the principles of free association. The third tool is that of listening. The physician attempts to pick up the cues and hints of what maybe going on inside the patient.

The last tool is to reflect back these cues to the patient. It is in this way that the interviewer is able to direct the interview. This method allows the physician to get around the defense mechanisms of denial and repression seen in hypochondrial patients.

The above method is diagnostic in the sense that it tells what is going on, it tells how serious it is, and it tells whether it may be related to the presenting symptomatology. It is therapeutic in that it allows the patient to ventilate some of the feelings or innter conflicts he may be having. If the doctor attempts to play the protagonist with the hypohcondriac-patient he is met with a wall of denial and hostility since he attacking the patients defenses against anxiety. The patient reacts by feeling that he is not being taken seriously, i.e., that his symptoms do not exist.

I feel there are also a number of ways of minimizing the risk of psychological probing. The first is not to allow too much material to come out too quickly. This may frighten off the patient and may bring out more material than the physician can handle at once. The second caution is not to interpret the connection between the inner-conflects and the hypochondrial symptomatology. This is a connection the patient must make for himself, either emotionally, intellectually or more important behaviourally, i.e., by no longer coming and proposing the symptoms. The aim of the therapy is to get the game between the doctor and the patient out in the open and theereby break the hypochondrial cycle.

I feel that the job up to this point is really under the auspiees of the general

practitioner. No one else is really in the same position to see the patient present initially untampered by investigation. Beyond this, depending on the nature of the conflicts, the kind of psycho-therapy needed, and the interest of the general practitioner further intervention may be decided upon. The patient, feeling that he has been taken seriously and is able to communicate with the doctor, is in a much better position for a psychiatric referral.

This approach is really an axample of preventive medicine. A general practitioner must get the hypochondrial patient in his unorganized state. For the patient who has been in an organized state for a number of years his neurosis has become to him a way of life, built into his personality structure and inter-personal relationships. If intervention occurs early, however, it may be able to break the cycle and save a great deal of time, money, and frustration for the patient and the doctor.

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The Unconscious Diabetic

SUMMARY

A rapid accurate diagnosis is essential when faced with a patient in diabetic coma. This article describes differential diagnoses, gives a flow chart of procedures to follow in diabetic coma, outlines methods of therapy and warns against the pitfalls attendant upon this condition.

Students involved in the curriculum changes taking place in most medical schools are enjoying a progressive exposure to clinical medicine, beginning in their first year. This provides the student with early insight and motivation in regard to his eventual role as a clinician. This alteration in teaching has removed much of the boredom of a full day of lectures by introducing the challenge of the bedside. This challenge provides many anxious moments for the clinical neophyte, previously accustomed to the orderly approach of the textbook or lecture. As the student learns to extract a useful story from his patient, elicit physical findings and edit a meaningful report, he more fully appreciates the sciences basic to clinical medicine.

Medical emergencies are a source of anxiety for even the experienced clinician. The better grasp the clinician has of the pathophyiology relevant to acute medical situations, the greater confidence he has in providing the acutely ill patient with adequate support. The unconcious diabetic is a good example of a medical emergency where an understanding of basic pathophysiology allows an orderly and effective approach to a challenging clinical problem.

In Medical emergencies the physician must represent decisive leadership in a situation of potential panic. Leadership will be exemplified by calm assessment of the patient's problems and an integrated approach to them.

In the severely ill diabetic where several clinical problems interrelate, rigidly memorized schemes for patient treatment often become illogical and even dangerous if instituted without understanding the pathophysiology of the imbalance. A simple and orderly approach to management follows logically from review of the pathophysiology.

Pathophysiology

Insulin plays a central role in body anabolism. It is the major fuel storage hormone, promoting the efficient uptake and utilization of glucose by many body cells. In adipose tissue, insulin promotes the buildup of triglyceride and also inhibits the separate pathway of lipolysis which leads to release of free fatty acids.

Endogenous insulin deficiency (diabetes mellitus) results in inefficient glucose metabolism and glucose wasting. Hyperglycemia develops and renal excretion of this excess serum glucose causes an osmotic diuresis. This eventually leads to depletion of body stores of carbohydrate (glycogen) and precursors of carbohydrate (Protein). The clinical picture therefore consists of polyuria and thirst with polydipsia, progressive weakness and weight loss. If the endogenous insulin deficiency is severe, this catabolic process is accelerated and extends to include the mobilization of large amounts of free fatty acids from adipose tissue stores. These free fatty acids are split into two carbon acetyl fragments in the liver, which in turn result in the formation of large amounts of ketones (acid radicals produced from neutral lipids). These acids are buffered by depleting bicarbonate and other body buffers, resulting in a progressive metablic acidosis. In ketoacidosis, coma is due to acidosis and dehydration.

In a few patients, often elderly or with compromised renal function, the carbohydrate intolerance results in marked hyperglycemia and dehydration, precipitating a crisis before significant lipid catabolism occurs. Coma is due to dehydration of brain cells and compromised organ perfusion due to shock. This syndrome is called non-ketotic hyperosmolar coma.

Some patients, usually elderly or with compromised renal function and usually receiving biguanide therapy, develop a selective failure of oxidative metabolism in many body cells. The resulting tissue hypoxia is manifest chemically as lactic acidosis. Coma is thought to be due to acidosis, hypoxia and dehydration.

Diagnosis

As coma in the diabetic may or may not be related to the intrinsic metabolic defect, its occurrence calls for rapid, accurate diagnosis, since treatment must be prompt and specific. The causes of coma can be grouped as follows:

1. **Related to diabetes and its treatment.**
   Hypoglycemia (treatment induced)
   Ketoacidosis
   Non-ketotic hyperosmolar coma
   Lactic acidosis
2. **Diabetes associated.**
   Uremia
   Cerebrovascular accidents
3. **General Causes**
   Infection
   Drug overdose
   Neurologic lesions

Speed in establishing the diagnosis and instituting appropriate therapy is most important, since mortality in the first group of patients is directly related to the duration of time before treatment. In the known diabetic there is generally little difficulty in diagnosis, the commonest by far being hypoglycemic coma. Symptoms at onset of coma, speed of onset and results of simple blood and urine tests will differentiate the first group. To assist rapid diagnosis, one should be aware of syndromes causing coma in the diabetic and the setting in which the various types will occur. In particular, alternate explanations are all too often given for abdominal pain, vomiting, hyperpnea and drowsiness occurring in the diabetic ill for several days, and correct treatment is all too often delayed. Where metabolic acidosis is recognized and ketosis is minimal or absent, the clinician must consider the possibility of lactic acidosis, as well as uremia or ingestion of acid such as salicylate.

Sudden onset of coma in a previously well patient who takes insulin or oral hypoglycemic therapy should arouse suspicion of hypoglycemia. In cases of rapid decrease in the blood glucose level, the body's homeostatic mechanisms effect a release of catecholamines which produce the signs and symptoms of tachycardia, sweating, pallor, tremor and hunger. In severe hypoglycemia CNS function is profoundly altered, and the individual may display symptoms ranging from headache, change in affect, anorexia, irritability or lethargy to those of psychotic behaviour, seizures or frank coma. Occasionally, severe hypo- or hyperthermia have been associated with hypoglycemia.

Therapy

First establish the diagnosis. If there is any uncertainty, administer 50 percent dextrose in water (about 50 cc or 25 g) after collecting blood for initial glucose and chemistry studies. Dextrostix determination is a useful rough guide. After drawing the initial blood tests establish an intravenous line. If the patient is elderly, hypo- or hypertensive or known to have cardiac disease, a central venous catheter should be inserted.

Blood Chemistry Studies:

1. Blood (serum) glucose; venous bicarbonate, sodium, potassium and chloride ions; BUN; hematocrit and white blood cell count.

2. Serum ketones are quantitated with acetest tablets[1] (the Rothera reaction). This measures acetoacetate and acetone but not B-hydroxybutyrate, which may be the predominant ketoacid if significant tissue hypoxia prevails, as in severe dehydration. B-hydroxybutyrate and lactate produce significant anion gaps in the absence of significant ketosis (serum ketones strongly positive in at least a 1:4 dilution). A serum lactate can be estimated if indicated. During therapy the serum ketones may paradoxically appear to increase, since the improved tissue perfusion with fluid replacement is causing conversion of B-hydroxybutyrate to acetoacetate.

3. Arterial pH, pCO_2, pO_2 and base excess.

4. Serum osmolality may be determined by direct measurement or by calculation as follows: 2 (Na + K) + blood sugar/18 + BUN/2.8. Normal osmolarity is 280 ± 10 mOsm per liter.

5. Blood culture and other appropriate cultures such as urine, sputum, nose and throat, CSF, etc.

Establish a flow sheet recording pertinent data, patient's vital signs and response. At the top of the flow sheet should appear a list of the clinical problems, e.g.:

Patient's name, age and diagnosis.

Problems:

1. Hyperglycemia
2. Dehydration and hypotension
3. Electrolyte imbalance
   a. acidosis
   b. potential hypokalemia with therapy
4. Urinary tract infection
5. Coma

Flow Sheet:

1. Urine glucose and acetone every hour
2. Blood (glucose, electrolytes, pH and BUN) every two to four hours
3. Serum ketones every two hours as a substitute for arterial pH
4. Intake and output hourly
5. Vital signs every hour as necessary

Insert a Foley catheter only if the patient is unable to void.

Fluids and Electrolytes:

If hypotension or shock is present, rapidly administer a volume expander such as dextran, albumin or whole blood and, if necessary, pressors such as metaraminol (Aramine) or norepinephrine.

Fluid Replacement:

If there is no hypotension, and depending on the degree of dehydration, age and cardiovascular status, begin infusion of 0.45 percent saline at a rate of 500-1500 ml per hour in an average sized adult.[2] The first liter can be given in 30 minutes and the above intravenous rate should be continued for the first two to five hours. Once the initial blood chemistry results are available, alter the fluid prescription accordingly. If hyperosmolarity is not present, isosmolar fluids are generally used. If hyperosmolarity is present but dehydration has resulted in significant hypotension, then isosmolar solutions must be given until there is no further danger of shock.

If congestive heart failure develops and central venous pressure rises excessively, the volume infused should be reduced. When the blood sugar falls, hypotension and oliguria may occur in some patients who are very dehydrated, despite infusion of apparently adequate fluids. This denotes latent dehydration and shock, requiring infusion of a volume expander.

Alkali Infusion:

Alkali is not necessary in the treatment of ketoacidosis and its use can be very dangerous if hypokalemia threatens. Alkali may be given for severe ketoacidosis, pH 6.9-7.0; in the presence of lactic acidosis; if severe hyperkalemia is present; or if the patient is in deep coma, severe hypotension or shock unresponsive to treatment. The use of bicarbonate in metabolic acidosis can create an imbalance in red blood cells between 2, 3 DPG (diphosphoglycerate) and pH which will adversely affect the ability of hemoglobin to release oxygen to tissues.[3] Phosphate, if available, can be given as the potassium salt to assist in restoring 2,3 DPG.

Potassium Infusion:

Potassium is generally depleted by catabolism and diuresis prior to onset of coma. The degree of dehydration will affect the concentration of serum K^+ such that it may be low, normal or high.

Recent studies have emphasized the enormous loss of K^+ in diabetic ketoacidosis and hyperosmolar coma, and the frequency with which hypokalemia develops. As treatment progresses, further massive loss may occur by entry of K^+ into cells, as well as renal excretion.

Once adequate urine flow is established, potassium should be added to the intravenous solutions in amounts indicated by repeated electrolyte determination (see flow sheet). If the initial serum K^+ is four or five mEq/liter, administer 20 mEq k/hour; if K^+ is less than four mEq/liter initially, infuse K^+ at the rate of 40 mEq/hour for two to three hours, then 20 mEq/hour depending on subsequent serum K^+ determinations. If K^+ falls to less than 3.5 mEq/liter by the end of the first hour of treatment, give at least 40 mEq K^+/hour, with 60 mEq k^+/hour if the serum K^+ is less than 2.5 mEq/liter. When giving large amounts, potassium phosphate or acetate will avoid the hyperchloremia which may develop in some of these patients due to excessive infusion of chloride. For every rise or fall of 0.1 pH unit, there is an average fall or rise of 0.63 mEq of K^+ per liter of plasma. This shift readily explains the danger of inducing severe hypokalemia (with attendant cardiac arrest) by rapid correction or acidosis without close attention to repletion of body K^+.

Five percent dextrose in water can usually be given by the third to fifth hour, whenever clear evidence of a progressive fall in blood sugar occurs.

Insulin [crystalline]:

Initially, short-acting crystalline insulin should be given intravenously. This will permit rapid action, allowing further frequent changes in insulin dosage as indicated by blood sugar determinations. Administer 0.25-1.5 units/kg of ideal body weight initially; larger doses are reserved for severely acidotic comatose patients. Depending on the response, as indicated by the state of consciousness, blood pressure, pH, serum acetone, blood sugar level, etc., subsequent doses of 0.25-0.5 units per kg may be given every one to four hours. Low dose insulin regimens have been reported recently — one by Genuth [4] for continuous low dose intravenous insulin, and one for intramuscular insulin on an hourly basis by Alberti, Hockaday and Turner.[5]

If the response to insulin is very rapid, further insulin should be temporarily withheld and five to ten percent dextrose in water infused until the blood sugar rises. 'Resistance' to insulin with persisting high blood sugars after four to six hous of therapy can be treated by doubling the dose of insulin administered every hour or even half hour. However, there is no convincing evidence that increasing the dose of insulin in this situation results in a beneficial overall response.

Gastric lavage should be performed on all ketoacidotic, comatose or hyperosmolar diabetic patients. If comatose, they may require endotracheal intubation first to prevent aspiration. Even slight elevations in body temperature warrant a vigorous search for infection, including lumbar puncture.[6] Meningitis especially must be considered in the comatose diabetic patient. After appropriate cul-

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A Study of Executives with Type A Behaviour

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In 1971, during the designing of the multi-centre Ontario Exercise Heart Collaborative Study of recurrent myocardial infarction, it became apparent that risk factors related to recurrent infarction would have to be stratified to ensure equal distribution in the two experimental groups (1). One of these risk factors was the presence of the so-called Type A Behaviour (2, 3).

Type A individuals are characterized by chronic and excessive struggle, competitiveness, ambition and impatience. They also exhibit, in an overt or repressed form, a high need for achievement, a strong sense of time urgency and a tendency toward chronic conflict with one or a group of persons, either by preference or necessity. This behavioural syndrome was first described by Friedman and Rosenman (4,5,6) and is identified using a specific interview format.

In order to permit identification of the Type A pattern in subjects entering the Ontario Exercise Heart Collaborative Study, Dr. Rosenman visited this centre and trained several interviewers. One of the authors (J.H.H.) of this paper had become deeply interested in managerial stress and the incidence of Type A Behaviour among executives, and took part in the teaching interview sessions with Dr. Rosenman and the other authors of this paper (P.A.R. and D.A.C). This was the beginning of a merging interest among the three authors, of vocational stress in clinically healthy individuals and its relationship to a number of variables including serum lipids, exercise habits, body fatness, etc. We subsequently have studied the relationship of fitness and stress among dentists (7), firemen (8) and executives (9,10). This paper describes some base line health data in a five year longitudinal study of executive stress. It concerns itself with Type A Behaviour and examines its relationship to certain measures of health and fitness among a group of 236 managers.

METHOD

Managers from 12 different Canadian companies were examined for the prevalence of Type A Behaviour. The data collected is part of a five-year longitudinal study of management stress. Overall, the data include a number of measures of health, measures covering issues of job and career, and measures of personality, life change, and family environment. Measurements include resting blood pressures, serum lipids (cholesterol, triglycerides), uric acid levels, and an exercise stress test. The data presented here relate specifically to the prevalence of Type A Behaviour and the relationships to a number of health indicators.

ORGANIZATION PATTERNS OF TYPE A BEHAVIOUR

Using the interview technique, the participants were classified into one of four behavioural categories according to the original typology developed by Rosenman and Friedman. The categories are entitled A_1, A_2, B_3, and B_4. The A_1 and B_4 categories represent fully developed or extreme Type A and Type B Behaviour.

Approximately 60 percent of the managers responding were classified as Type A's. Twenty-seven percent were classified as the extreme Type A_1. Only 12 percent of the respondents were fully developed Type B_4. These percentages are similar to those reported by Rosenman (5) who observed that in 1960-61 nearly half of his 3500 male subjects exhibited Type A Behaviour. Rosenman has speculated that the frequency of Type A Behaviour has increased significantly since 1960-61 (now as high as 75 percent) due to greatly increased socioeconomic pressures and that, in general, Type A Behaviour has been increasing in industrialized societies.

Table 2 showing the percentage of Type A/Type B Behaviour in 11 of the 12 companies studied supports these speculations. Among these 11 companies the percentage of Type A Behaviour varied from 50 to 76 percent. Type A_1 Behaviour varied from 13 percent to 43 percent. This variance in Type A Behaviour between companies was related to the company's recent growth rate. In the five companies with the highest growth rates, 66 percent of the managers were classified at Type A. In the five companies with the lowest growth rates, only 56 percent of the managers were classified as Type A ($p = .07$).

Age and education were examined with regard to their influence on Type A Behaviour. There were no significant trends in terms of education level, although there was a slight indication that lower levels of education were associated with a greater prevalence of Type A Behaviour.

Type A Behaviour declined slightly with age and Type B Behaviour increased slightly with age. It was noticeable that the highest percentage of Type A_1 Behaviour occurred in the age group 36 to 55 years - peak years in which many issues of job and career take on a sense of urgency.

TYPE A BEHAVIOUR AND HEALTH

Several measures of health were used in examining differences between Type A and Type B individuals. These included a symptom checklist, blood tests, exercise tests, and a survey of health habits. Table 3 presents the average number of stress symptoms reported on the symptom checklist by Type A and Type B individuals.*

Individuals classified as Type A_1 report significantly more symptoms than all other managers. In addition, all Type A's in comparison with all Type B's report more symptoms, but the differences are not as great. It would seem that the fully developed Type A_1's incur the greatest risk in terms of stress symptoms.

In addition to a simple counting of stress symptoms, the symptom checklist was factor analysed and five factors emerged. Based on the symptoms composition of each facter, these factors were entitled Cardiovascular, Emotional Distress, Insomnia, Gastrointestinal, and Diabetes.

The mean factor scores indicate whether a particular behavioural type tends to be high or low on the types of symptoms composing that particular factor. The scores are not significant in a statistical sense but provide interesting information. The mean factor scores on each factor for each of the behavioural types is shown in Table 4.

Most important is the finding that the Type A_1's tend to be high on each of the factors and that the biggest differences are between Type A_1 and Type B_4. Health differences appear to be particularly acute between the fully developed Type A_1 and Type B_4. On one of the factors - gastrointestinal problems - the difference between Type A_1 and Type B_4 is quite small. It would appear that in the case of gastrointestinal symptoms there are few differences between the Type A's and Type B's. The emotional distress factor which

*This symptom checklist is a composite list constructed with reference to previous research on stress symptoms. (12-18) In previous work by the authors the list has been used as an effective indicator of stress by both the simple addition of the number of stress symptoms reported and by the identification of health factors using factor analytic techniques. In table 3 a simple addition of symptoms reported has been used.

TABLE 1

Type A/B Behavior — Percentage of Respondents

Behavior Type	Number	%	
A_1	64	27%	61% A's
A_2	81	34%	
B_3	63	27%	39% B's
B_4	28	12%	
	n=236	100%	

TABLE 2

Type A/Type B Behavior by Company

Behavior Type	Low Growth Companies						High Growth Companies				
	1	2	3	4	5	6	7	8	9	10	11
Type A	58%	52%	50%	57%	60%	67%	65%	57%	76%	71%	62%
Type B	42%	48%	43%	43%	40%	33%	30%	43%	24%	29%	38%
N =	(24)	(44)	(14)	(23)	(20)	(24)	(17)	(14)	(17)	(14)	(24)

TABLE 3

Type A/Type B Behavior and Stress Symptoms

Behavior Type	Average Symptoms Reported During Past 12 Months	N
Type A_1	3.49	63
Type A_2	2.10	80
Type B_3	2.30	61
Type B_4	1.90	27
	$P(A_1)$=0.003	
	P(A/B)= 0.07*	

* The probability $P(A_1)$ is a one-sided large sample theory test of the difference between two means. $P(A_1)$ tests the difference between the A_1's and all others where the null hypothesis is that there is no difference. P(A/B) tests all Type A's (A_1's and A_2's combined) against all Type B's (B_3's and B_4's combined). This notation is used throughout the paper.

TABLE 4

Mean Factor Scores

	Factors				
Type	Cardiovascular	Emotional Distress	Insomnia	Gastrointestinal	Diabetes
A_1	.20	.22	.16	.13	.16
A_2	−.03	−.10	−.05	−.15	−.05
B_3	−.02	−.05	−.01	−.01	−.05
B_4	−.14	−.16	.01	.06	−.07
	$P(A_1/B_4)$=.08	$P(A_1/B_4)$=.03	$P(A_1/B_4)$>.10	$P(A_1/B_4)$>.10	$P(A_1/B_4)$>.10

TABLE 5

Blood Analyses — Mean Values

Type	Systolic Blood Pressure	Diastolic Blood Pressure	Cholesterol	Triglycerides	Uric Acid	(N)
A_1	144	88	240	219	6.4	(56)
A_2	132	83	225	180	6.5	(78)
B_3	130	82	230	172	6.6	(59)
B_4	131	80	232	185	6.3	(27)
	$P(A_1)=.0001$	$P(A_1)=.004$	$P(A_1)=.02$	$P(A_1)=.04$	$P(A_1)=NS$	
	$P(A/B)=.001$	$P(A/B)=.04$	$P(A/B)=NS$	$P(A/B)=.10$	$P(A/B)=NS$	

TABLE 6

Type A/B Behavior and Cigarette Smoking

Type	Percent Who Smoke	Average Cigarettes/Day (of Smokers)	N
A_1	41%	25	64
A_2	25%	20	81
B_3	25%	25	63
B_4	29%	23	28
			N=236
	$P(A_1)=.02$		
	$P(A/B)=NS$		

TABLE 7

Exercise and Predicted Max. Oxygen Uptake ($\dot{V}O_2$) (ml/Kg.min.)
By Age Groups

Do you make a specific attempt to get a minimum amount of exercise?	0-30	31-40	41-50	51-55	56 up
Yes	39.4(9)	34.8(36)	30.6(29)	30.22(15)	25.9(14)
No	32.9(8)	32.5(20)	28.6(26)	29.6(6)	21.6(5)
	(p=.05)	(NS)	(NS)	(NS)	(p=.10)
(N)	(17)	(56)	(55)	(21)	(19)
Percent exercisers	53%	64%	53%	71%	74%

TABLE 8

Type A/B and Exercise
Percent Exercisers by Age and Type A/B

	0-30	31-40	41-50	51-55	56 up
Type A's	46% (11)	64% (33)	49% (37)	69% (13)	100% (7)
Type B's	67% (6)	65% (22)	61% (18)	75% (8)	58% (12)
N =	(17)	(55)	(55)	(21)	(19)

shows the only significant difference, included symptoms such as restlessness and agitation, fatigue, depression, the need to be left alone, difficulty in concentrating, and high use of tranquilizers.

BLOOD ANALYSES

A sample of blood was taken from each of the participants and examined for serum cholesterol, triglycerides and uric acid. In addition, blood pressure was taken for each individual. The results by behaviour type are shown in Table 5.

Although the absolute differences are small, on all measures except uric acid, the Type A_1's are significantly higher than all others. In addition, on systolic and diastolic blood pressure and on serum triglyceride levels, all Type A's are higher than Type B's. On serum uric acid there appears to be little difference in terms of behaviour type. These results tend to confirm the findings of Friedman and Rosenman and are an indication of how the Type A Behaviour pattern is related to a number of risk factors in coronary heart disease, (5) but it is interesting to note that for both lipids the B_4 group in this sample was second highest.

SMOKING

As a part of the general health inventory the respondents were asked to indicate their exercise and cigarette smoking habits. A larger percentage of the Type A_1's were found to be cigarette smokers (Table 6). There was no significant differences, however, between all Type A's and all Type B's. In addition, of those who smoked, there were no differences between the types in the amount smoked.

EXERCISE BEHAVIOUR

A part of the testing of the participants included the calculation of predicted maximum oxygen uptake (VO_2) in (ml/kg/min) by use of the bicycle ergometer. (19) In addition, the participants were asked if they made a specific attempt to get a minimum amount of exercise. This question on exercise habits was then related to max. VO_2 by age groups and it was found that in every age group those who indicated that they made a specific attempt to get a minimum amount of exercise had a higher max. VO_2 (Table 7).

Type A/B Behaviour also was related to exercise habits (Table 8). It was found that in every age group except the oldest, a larger percentage of the Type B's were exercisers. In the oldest age group (56 years and up) all of the Type A's were exercisers (100 percent) while only 58 percent of the Type B's were exercisers.

Type A/B behaviour also was related to max. VO_2 by age group (Table 9). From Tables 7 and 8, the Type B's would be expected to have a higher max. VO_2. This was the case in the youngest age group (below 30), although the difference was not significant. In most age groups the difference in max. VO_2 between the Type A's and Type B's was very slight. This was not the case, however, in the oldest age group (56 years and up), in which the Type A's had a significantly lower max. VO_2 ($P = .05$). It is interesting that in the oldest age group all of the Type A's claim to be exercisers, but as a group they have the lowest max. VO_2. However, it should be noted that the number of subjects in this group was small ($N=7$), and therefore the relationship may be fortuitous.

It is possible that the Type A's lack of interest in exercise has the cumulative effect of resulting in a significantly lower max. VO_2 and that the change in exercise behaviour (after age 55) is a response to becoming aware of declining fitness. Type B individuals, on the other hand, may have maintained both their exercise habits and their fitness through most of their lives and carry a higher level of fitness into their older years. It is also worth noting that after age 55 the increased interest in exercise among the Type A's may not be rigorous enough to achieve a significant training effect and consequently improve their max. VO_2.

SUMMARY AND CONCLUSIONS

The data presented in this paper are part of that from a five-year longitudinal study on management stress. Approximately 300 top managers from 12 major Canadian companies are involved in this study. A part of this investigation is concerned with the patterns, prevalence, and causes of Type A behaviour, and we have presented in this paper trends on health variables significantly related to this behavioural syndrome. Many of these variables are known risk factors associated with the likelihood of coronary heart disease.

As an occupational group, managers, either by selection or consequence, would seem well exposed to job and career factors contributing to this behavioural syndrome.

Overall, 61 percent of the managers in the study were classified as Type A's. Between companies the prevalence of Type A Behaviour ranged from 50 percent to as high as 76 percent and, as might be expected, was found to be related to the recent growth rate experienced by their companies.

Trends in terms of age and education were not strong but indicated the possibility that lower levels of education and the early career years might be related to a higher prevalence of Type A Behaviour. The highest percentage of the extreme Type A_1's was found in the age group 36 to 55 years - important and active years for many issues of job and career.

The Type A_1's stand out as significantly different on the health factors, although on blood pressure (systolic and diastolic) all of the Type A's combined were significantly higher than all of the Type B's. The Type A_1's reported more stress symptoms, and they exhibited higher blood pressures, higher cholesterol, and higher triglyceride levels. There were no significant differences on uric acid. In addition, a larger percentage of the Type A_1's were cigarette smokers. Although the absolute differences on some of these values were not large, overall they indicate a distinct trend.

The Type A's reported less participation in exercise than the Type B's in each age group except the oldest (over 55 years). The cardiovascular effect (max. predicted VO_2) of this lower interest does not, however, appear significant until the age of 55 years.

In terms of coronary risk, the Type A_1's seem particularly well exposed. Blood pressure, smoking and serum lipids (cholesterol and triglycerides) are all high in comparison with the other groups. using the overall average values from the previous tables for the Type A_1's on these risk factors, and assuming the absence of left ventricular hypertrophy on the resting ECG and the absence of glucose intolerance, the Framingham data (20) predicts the probability of our average Type A_1 developing coronary heart disease in the next six years at 6.3 percent, based on these risk factors alone and not including whatever independent risk Type A carries with it. For the average Framingham man, age 45 years, this value is 4.4 percent and, consequently, shows our Type A_1's to have about a 50 percent greater risk. In this sense, our data predicts a higher prevalence of coronary heart disease among Type A_1's. With regard to the Type A_2's, the findings are not as conclusive, in that many of the risk factors were not significantly different from those of the Type B's. Our conclusion, consequently, is that only the extreme Type A_1's are significantly different on coronary risk, although we would add that a more demanding job environment would have its greatest influence on moving Type A_2's toward Type A_1's.

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The Unconscious Diabetic

(cont'd from page 12)

tures are obtained, if infection is suspected, it should probably be treated, preferably with penicillin. If a specific infection is found, appropriate antibiotic therapy should be used. A chest X-ray should be a part of the initial workup.

Once the patient is awake and able to take fluids, remove the nasogastric tube, IV tubes and the urinary catheter. The patient can usually be started on his intermediate acting therapy as soon as blood sugar is controlled and he is taking oral fluids. If control lapses again, this should be treated acutely by the physician, not by the nurse on a 'coverage' basis (eg: insulin by urine sugar results).

Pitfalls to be Considered

1. Coma in the diabetic can be caused by all the usual etiologies of coma seen in non-diabetic patients — e.g. hypolglycemia, uremia, stroke, overdose, skull trauma, etc.

2. Abdominal pain of ketoacidosis may mimic an acute condition of the abdomen requiring surgery. However, an acute abdominal condition can occur in the diabetic patient, and a missed diagnosis could be disastrous. Consider acute appendicitis, empyema of the gall bladder, and acute pancreatitis.

3. Pleural and pericardial friction rubs and pain may occur in the severely dehydrated patient. If these findings do not resolve quickly on rehydration, consider uremic or viral pericarditis, myocardial infarction or pleurisy. On the other hand, in the severely dehydrated patient, rales and other physical findings of pneumonia may not be evident until rehydration, a reminder that repeat physical examination during management is indicated.

4. Urinary tract infections frequently precipitate ketoacidosis, often with a background of atonic bladder.

5. Low serum sodium concentrations in diabetic coma may be secondary to:

a. Markedly elevated blood sugar with its osmotically obligated water, resulting in relative dilution of the Na^+.
b. 'Pseudohyponatremia' due to a marked evelation of the serum lipids, usually with grossly lactescent serum.
c. Less commonly, true hyponatremia with Na^+ loss in excess of water.

6. Sustained hypotension after the use of plasma volume expanders and pressors may point to a number of complicating factors. While evidence is weak, acidosis may be associated with persistent peripheral vasodilatation or impaired cardiac contractility. In persistent hypotension, cautious use of bicarbonate, usually with K^+, may be indicated. Other factors to be considered include myocardial infarction, gram-negative septicemia, staphylococcal or pneumococcal infection, renal papillary necrosis, hemorrhagic pancreatitis, gastrointestinal hemorrhage, or acute adrenal or pituitary insufficiency.

7. Edema or excessive weight gain due to fluid retention, may occur during vigorous treatment of diabetic ketoacidosis or hyperosmolar coma. This may be due to:

a. Excessive fluid and electrolyte replacement.
b. Cardiac, hepatic or renal failure.
c. Peripheral vascular insufficiency, which is common in diabetic patients.
d. Other causes, such as hypoalbuminemia, local obstruction to lymphatic or venous return, etc.

The problems seen early in the treatment of diabetic coma are related primarily to dehydration and shock, and less so to acidosis. The late problems relate primarily to hypoglycemia, which is much less common with a low dose insulin regimen. The preparation of a problem list and flow-sheet at the outset of therapy will permit an overview of the problems and of the response to an integrated therapeutic regimen.

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Class of '76 Internships

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Letters

Dear Sir:

Your readers will have learned, with sorrow, of the untimely and unexpected death of Dr. Roger J. Rossiter, Professor of Biochemistry and Vice-President (Health Sciences), sometime Provost and Vice-President (Academic) of the University, Dean of Graduate Studies and for many years (1947-1965) Head of the Department of Biochemistry. He died in Helsinki, Finland, on February 21st, 1976 while on study leave and was engaged in an inquiry concerning the teaching of medicine and primary health care on behalf of the Faculty. This brought to an end a remarkable and productive career as a scientist, teacher and administrator; a career that has done honour to both Roger Rossiter and Western. Some thiry classes in Medicine, a flock of graduate students and colleagues have good reason to temper sorrow with happier remembrances of times past, of more relaxed occasions, of moments of fun, of kindness and of good deeds performed on behalf of "the School" and its denizens.

Some who read this, and I hope that it will be a considerable number, will wish to join his many friends in perpetuating the generosity of his heart by contributing to **The Roger J. Rossiter Memorial Fund** (c/o The Comptroller of the University). The intent of his family and friends is that the fund provide substantial assistance to a worthy Post-Doctoral Fellow and President D.C. Williams has set-up an appropriate Committee to guide and administer the fund. A substantial and continuing award of this character will be a most appropriate memorial to the spirit and interests of our good friend and teacher.

Yours sincerely,

R.G.E. Murray, M.D.
Professor

To Class of '76
Dear Brontosaura:

You have been somewhat concerned by the problem of curriculum reform that left you as the last class to graduate from the old curriculum. You are now about to embark upon the practice of medicine; changes in that area will rid you of any concern that you have not experienced change and reform.

Now, as never before, the profession is beset by external controls. The realization by government that universal health insurance is extremely expensive has created a demand on the part of government for control of these costs. A large bureaucracy is being established to monitor physician accounts; fortunately in Ontario part of this review process remains in the hands of the profession. In the matter of manpower, increasing controls are appearing, at first with respect to immigration, but later, perhaps you may be restriced by local health councils in the number of practitioners allowed in any one area. The process of peer review is gathering momentum; re-licensure requirements may demand yet another external body to review your competence at intervals.

The point of this litany is simple. The physician of the future (you) may well be exposed to more controls and regulations than at any time in the past; to ensure some equality in any confrontation with these future bureaucracies the individual needs support. That support comes from your own medical societies. I take this opportunity to strongly urge you to join and participate in the local academies and medical societies, the provincial medical associations, and to continually inform your provincial licensing body of your opinions and concerns.

Finally, may I once again thank the class for the great honour it has bestowed upon me. I look forward to seeing all of you at the graduation ceremonies, many of you over the next few years during your internship and residency training programs and all of you again at your fifth year reunion in 1981.

Yours sincerly,

D.C. Bondy, M.D., F.R.C.P.[C]
Professor of Medicine
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